Introduction to Brain & Behaviour
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Lecture 28
Emotions and Other Cognitive Processes

Hello and welcome to the course, Introduction to Brain and Behaviour. I am Dr. Ark Verma from IIT, Kanpur. We are in the sixth week of the course and today we will continue to talk about emotions and their influence on other cognitive processes.

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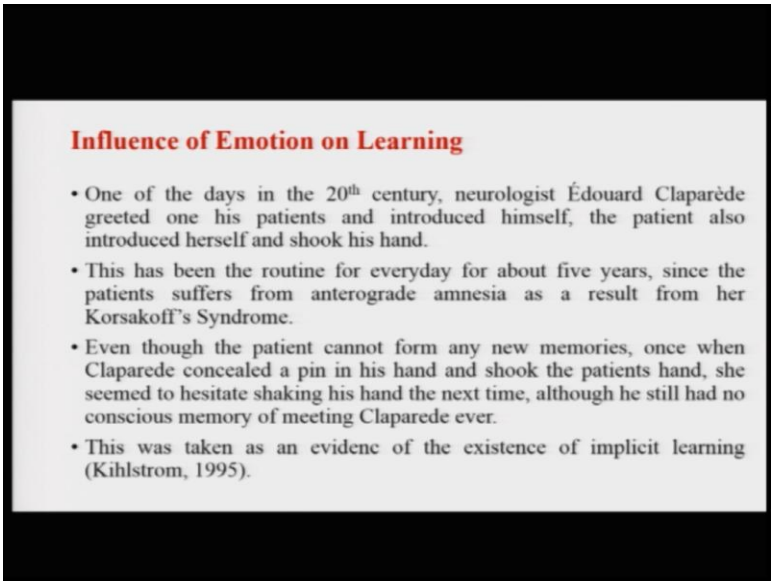

Let us talk about the influence of emotions on learning. How do we gain information? How do we pick up more information? How do we learn new skills, information, knowledge? And how does our emotional state or how do our emotions influence this? One of the day, on one of the days, and let me begin, let me begin talking about this with a bit of an anecdote.

So, on one of the days in the twentieth century, neurologist Edouard Claparede greeted his, greeted one of his patients and he introduced himself and the patient also introduced herself and they shook hands. Now this, however, has been going on for routinely for around 5 years. Because the patient has anterograde amnesia as a result of her Korsakoff syndrome. So, she does not remember, she does not realise that it is the same person. She has no memory of the doctor visiting her after a few hours.

Even though the patient cannot form any new memories, once what happened was that Claparede concealed a pin in his hand and he shook the patient's hand. Next day when he again came back and wanted to shake the participant's hand, the participant's although had no memory of seeing Claparede before but she hesitated in shaking his hand. This basically can be taken as an evidence of the exist, of the existence of implicit learning.

Even though explicitly the patient does not really remember Claparede or he does not remember having met him ever before, but the, the hesitation that the patient showed in shaking his hand, basically it is characteristic of the fact that somehow the experience of shaking hands when, the previous day when there was a pin in this person's hand has been there. It has somehow been registered. It has somehow been retained and that is why the next time the neurologist comes in, the person sort of, you know, hesitates in shaking hand which was the usual thing, which was going on for over 5 years.

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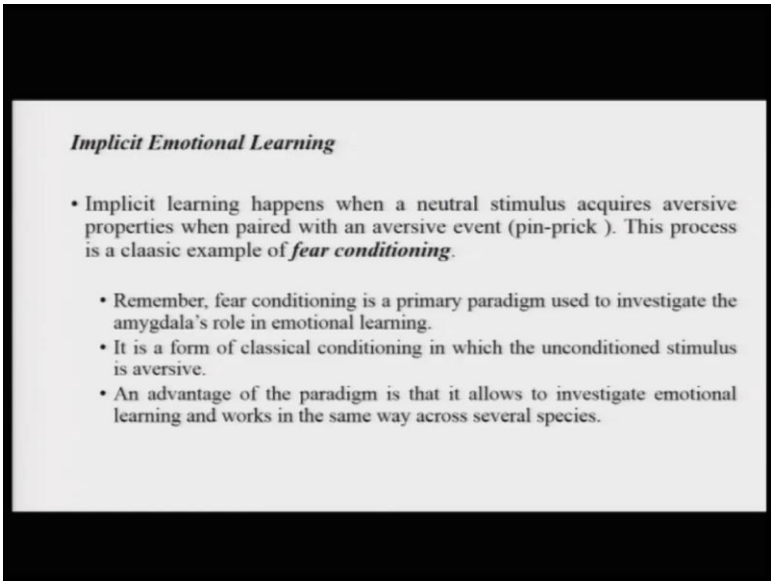

So, this is basically what is called implicit emotional learning or implicit learning which has some influence of the emotion. Let us see. Now, implicit learning as you have seen, happens when a neutral stimulus acquires aversive properties when paired with an aversive event. So basically, this process is a classic example of fear conditioning. Remember, we were talking in the last chapter about fear conditioning.

Fear conditioning is when say, for example, a particular animal is conditioned to fear a particular situation or an event. Fear conditioning is a primary paradigm that is used to investigate the amygdala's role in emotional learning. It is a form of classical conditioning in which the unconditioned stimulus is aversive.

So, the stimulus with which the conditioning is triggered is basically is an aversive stimuli which will base, which will automatically invoke, you know, unpleasant or aversive response. An advantage of this paradigm is that it allows to investigate emotional learning and works in pretty much the same way across several species

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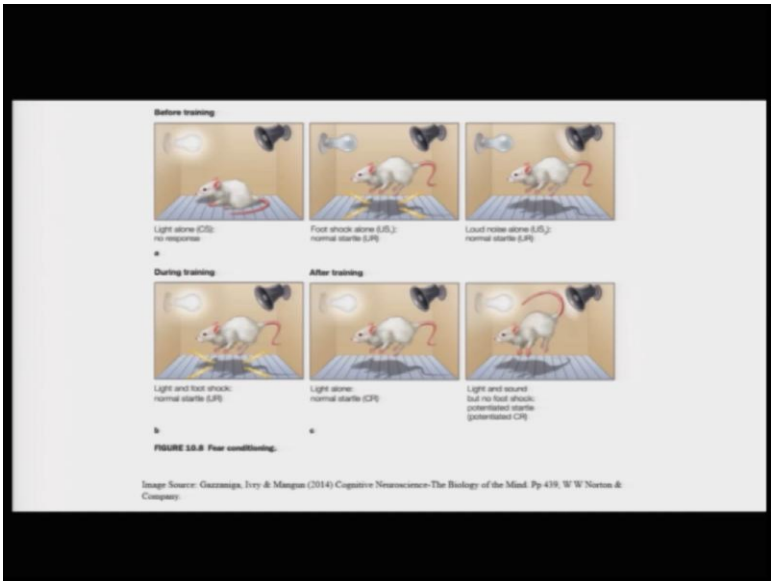

Let us take an example. Here you can see that before training, there is just the light alone and there is no response and the, you can see that the rat is in this chamber. Next time what you can see, there is this unconditioned stimulus of the foot shock. So, the foot shock basically is an unconditioned stimulus which is given to the rat and basically rat gets startled. You can see the rat is now in the air and it has gotten startled.

In the third slide you are seeing is that, as soon as the loud sound noise was presented, loud noise was presented, there is a normal startle. So, there are two things. One is the foot shock alone and the other is the loud noise alone. After training you can see that, if there is both light and foot shock together, the participant, the rat feels startled in the normal fashion. When there is right,

light alone, because light has been earlier paired with the electric shock, the participant, the rat again gets startled.

Similarly, if you can see that it is almost leading to second order conditioning as well. Say, for example, when light and sound are both presented but the foot shock is not presented, the rat is again startled. So basically, what is happening is that the rat is associating the light with the shock and in a later point, it is basically associating the sound and light together with the shock. So, this is just an example of how fear conditioning takes place.

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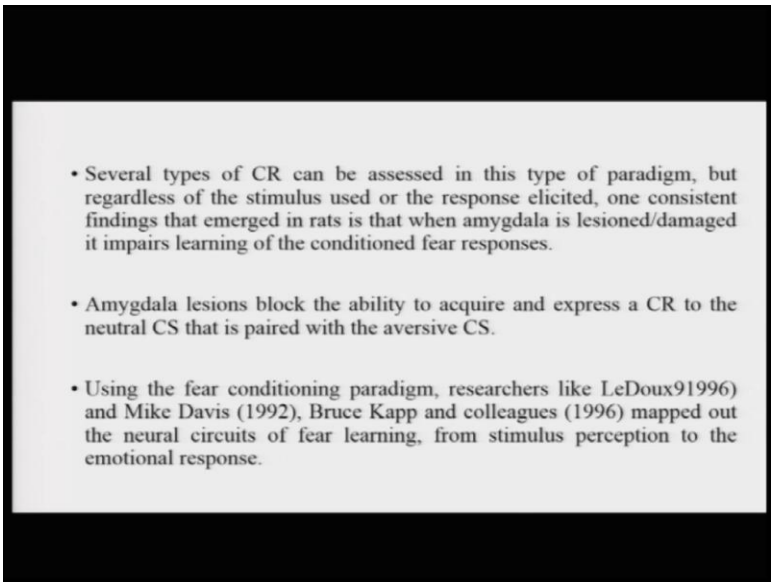

Now, several types of conditioned responses, that of getting startled, can be assessed in this type of paradigm. Regardless of, but regardless of the stimulus used or the response elicited, one consistent finding that has emerged in rats is that when the amygdala is lesioned or damaged, it impairs the learning of these conditioned fear responses. So, what will happen is that, if the amygdala is lesioned or damaged in these rats, they will not be able to associate the, the light and the shock and they will not show the conditioned fear responses of startling.

Amygdala lesions probably block the ability to acquire and express a conditioned response to the neutral (cana), to the neutral conditioned stimulus that is paired with the aversive stimulus. So next time the, the rat is going to hear this loud noise or there is this light, the rat will not freeze up. It will not get startled.

Now using the fear condition paradigm, researchers like LeDoux and Mike Davis, Bruce Kapp and colleagues have mapped out the neural circuits of fear learning from stimulus perception to emotional response. Let us look at that.

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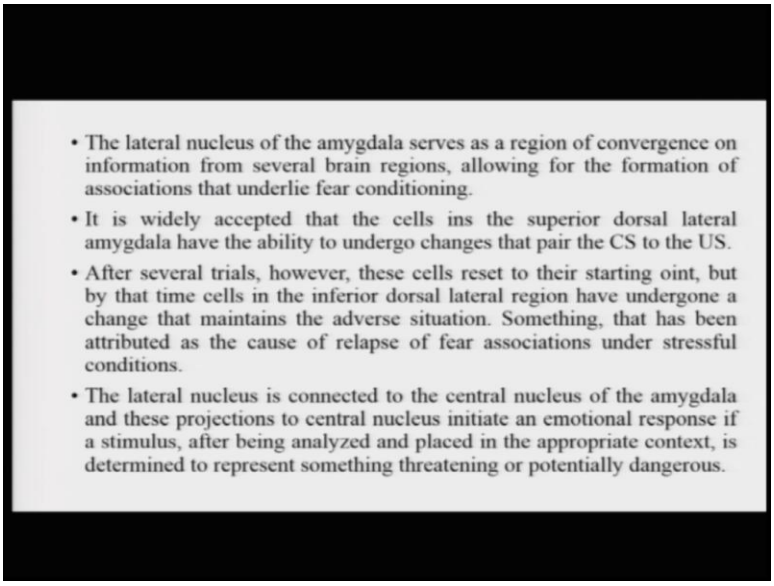

The lateral nucleus of the amygdala serves as the, as a region of convergence of information from several brain regions which allows for the formation of associations that underlie this process of fear conditioning. It is widely accepted that the cells in the superior dorsal lateral amygdala have the ability to undergo the changes that pair the CS to the US. So basically, if this region is not functioning properly then the association between the CS and the US that is the conditioned stimulus and the unconditioned stimulus might not be able to happen.

After several trials of conditioning, however, these cells reset to their starting point. But by the time the cells have sort of resettled, the cells in the inferior dorsal lateral region, remember earlier I was talking about the superior dorsal lateral region, so by, after several trial these, the cells of the superior dorsal lateral region have reset themselves. But by that time the cells in the inferior dorsal lateral region have undergone a change that maintains this adverse pairing, that maintains or let us say remembers this adverse situation.

Something, that has been attributed as a cause of the relapse of fear conditioning under stressful conditions. Say, for example, if say, for example, in, in, in, you know, several days later again

the same kind of thing is there and the rat is placed under extreme, you know, experimental stress, then the conditioned response can actually come back.

Now, the lateral nucleus is connected to the central nucleus of the amygdala and these projections to the central nucleus initiate emotional response if a stimulus after being analysed and placed in appropriate context is determined to represent something threatening or something potentially dangerous.

An important aspect of this fear conditioning circuitry is that information about the fear inducing stimulus reaches the amygdala through two separate but simultaneous pathways. So, the idea is that this information about the fear conditioning stimulus reaches the amygdala from two pathways.

What are the two pathways? One pathway goes directly from the thalamus to the amygdala without being filtered by conscious control. So, there is no control, it is almost automatic. Signals sent by this pathway are sometimes called the low road that reach the amygdala rapidly, although the information in this pathway is crude. Say, for example, this might be able to explain your initial reaction to fearful stimuli.

At the same time, sensory information about the stimulus is being projected through the amygdala via another cortical pathway which is sometimes referred to as the high road but which is much slower. So, the high road is much slower. It takes around 300 milliseconds in rat, probably lesser or more in humans, we are not sure. But the analysis of this stimulus is more thorough and complete. You are definitely sure what the stimulus is about.

Let us give you an example. Suppose, again I am, I have probably given this example in an earlier lecture. Suppose you are walking in your garden and suddenly you hear a slithering sound and you hear, and you see something, some movement in the grass. The initial response basically to that slithering sound or that rustling of grass will be to step away and probably think that there is a snake. That is probably being done by this pathway that is going directly from the thalamus to the amygdala.

However, if you pause a little bit, if you kind of give yourself that kind of time, if you are capable of doing that, the other pathway will immediately in a moment of, you know, in a matter of seconds will tell you that okay, no, this is probably not the grass. It is basically the, you know,

it is basically probably the hose pipe which you are using to water the plants or something like that. So, this is very interesting in the sense that there, there is an autonomous, there is an autonomous, automatic faster pathway and there is a slightly slower pathway.

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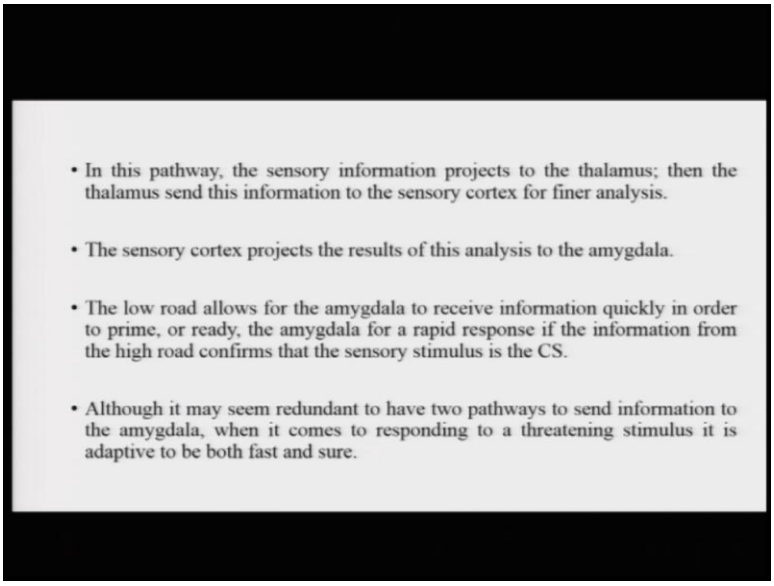

Now in this pathway, the sensory pathway that is, the sensory information projects to the thalamus; and then the thalamus projects this information to the sensory cortex for finally, for finer analysis. So, it goes through a more detailed analysis. Now the sensory cortex projects the results of this analysis to amygdala.

So, information flow is like a sensory information is directly coming to the thalamus. Thalamus is sending it to the higher sensory cortices. The sensory cortices are analysing that information in some detail and then they are projected, projecting the final results to the amygdala. The low road, on the contrary, allows for the amygdala to receive the information rather quickly in order to be primed or in order to be ready for a rapid response if the information in the high road confirms that the sensory stimulus is, let us say, the conditioned stimulus in this case.

Although it may seem redundant to have two pathways to send information to the amygdala, when it comes to responding a threating stimulus, it is adaptive because it makes you very fast and it makes and it basically tells you that, okay, you can take the time to be sure later. But if the stimulus is let us say a snake, it is better to take evasive action rather quickly and rather rapidly.

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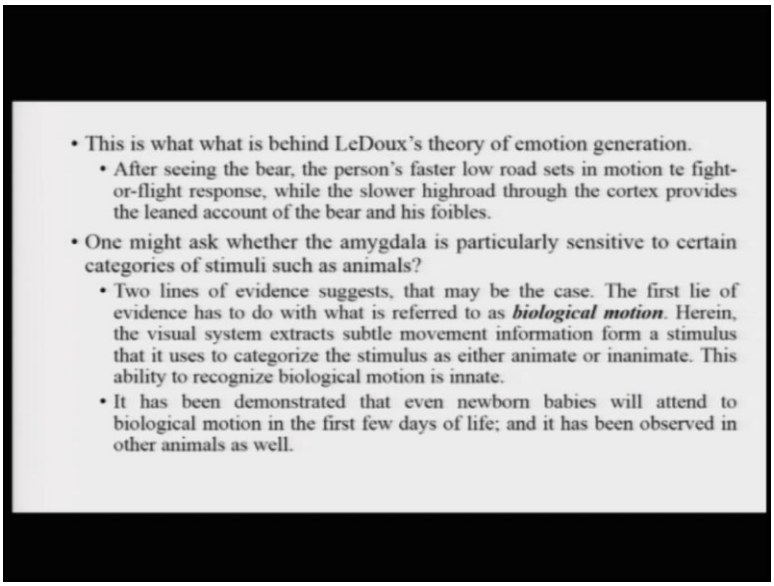

Now, this is basically what was, what might be the reason behind LeDoux's theory of emotion generation. After seeing the bear, the person's faster lower road sets in the, sets in motion the fight or flight response. You know that you have to run away from there or you have to, say, for example, if you are carrying a weapon or something, confront the bear. While the slower high road through, which is coming through the cortex provides the learned account of the bear and his weaknesses and whatever your options are and sort of gives you a slightly detailed plan of action.

Again, it both depends upon what, what is the time at hand. Is the bear approaching you with an intention to attack you? Or is the bear is just around and minding his own business and, but it still is, is still a cause for fear.

Now one might ask whether the amygdala is particularly sensitive to certain categories of stimuli such as animals. Two lines of evidence suggests that, that actually may be the case. Let us say, the first line of evidence has to do with what is referred to as biological motion. Now biological motion is basically the fact that when the visual system extracts subtle movement information from a stimulus that it uses to categorize the stimulus as animate or inanimate.

See we know that different stimuli are, you know, have different characteristics but movement is peculiarly the property of animate things, mostly living things that, you know, are there and move around us. This ability to recognize biological motion is innate. It is evolutionarily built-in

in our system. Now it is been demonstrated that even new born babies will attend to the biological motion in the first few days of their life and they are able to categorize objects as animate versus inanimate. Also, this ability has been found in some other animals at early age as well.

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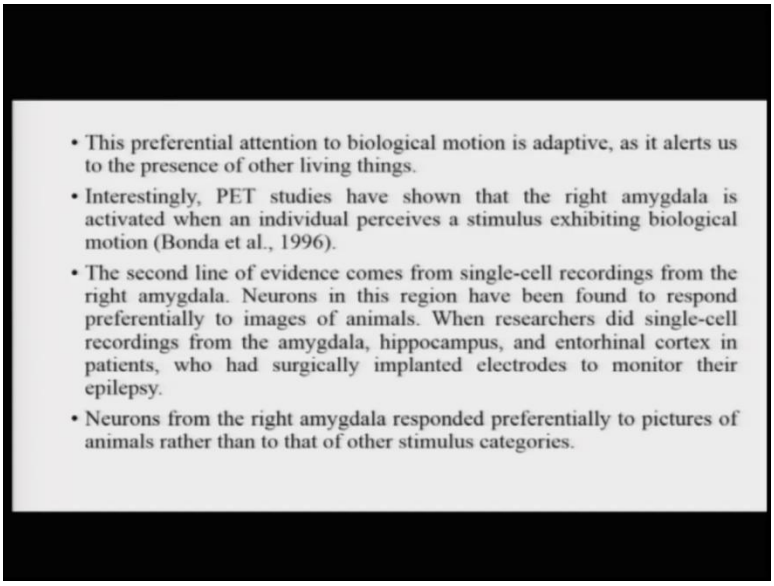

Now, this preferential attention to biological motion is adaptive, it has its own benefits. You should or you, it is very valuable to know if there are living things around us. Okay? Sometimes to take evasive action, sometimes to form societal relationships. Now, interestingly, Positron Emission Tomography studies have shown that the right amygdala is activated when an individual perceives a stimulus exhibiting biological motion, if basically, if a living object is moving around the person.

Now the second line of evidence comes from single cell recordings from the right amygdala. Neurons in this region have been shown to, have been found to respond preferentially to images of animals. Now we know that if you see images of animals there is a high probability of or say, for example, in the conceptual analysis of the pictures of animals, there is this thing of biological motion that you will automatically decipher.

Now when researchers did single cell recordings from the amygdala, hippocampus and the entorhinal cortex, in patients who had surgically implanted electrodes to monitor their epilepsy,

we have talked about this, now neurons from this right amygdala responded preferentially to pictures of animal, animals, rather than, rather than to that of other stimulus categories.

So, it seems that the right amygdala is sort of coding information which is relevant, a to biological motion and b to categorize objects into animate and inanimate categories.

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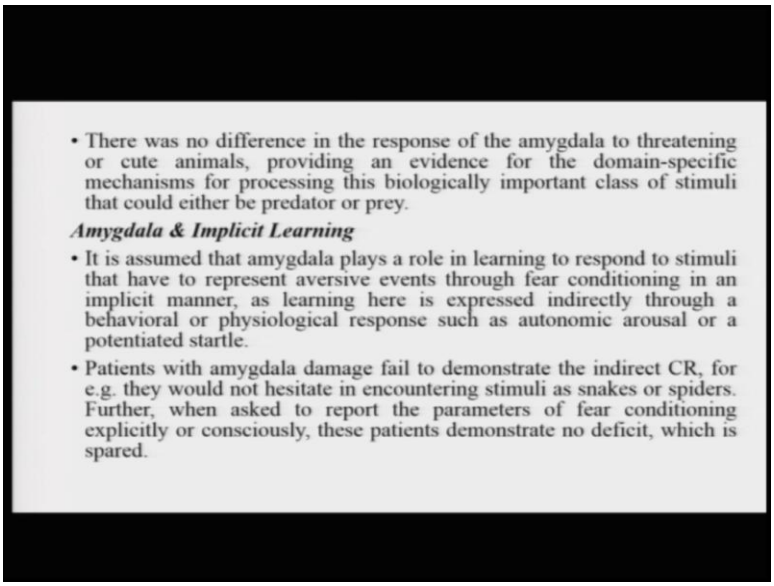

Now, there was no difference in the response of the amygdala to threatening or cute animals, which basically means that there might be a dedicated network that is basically, you know, specialized or it is domains specific, is using a domain specific mechanism to basically categorize animate and inanimate things at this moment but not really whether they are cute or say, threatening for that matter. So, there is a bit of a, there is a bit of a domain specificity here.

Now what is the role that the amygdala might play in implicit learning. Let us look at that in some detail. Now it is assumed that the amygdala plays a role to respond, in learning to respond to stimuli that have to, that have to represent aversive events through fear conditioning in an implicit manner. So, it is not explicit.

It is in an implicit manner that if there is a particular stimulus around you which, you know, represents a potential fearful, you know, aspect. Say, for example, if there is a, you know, if you are afraid of dogs, okay, and you are around a friend who has a pet dog and the friend loves the dog. The dog loves the friend. Everything is alright. But you have let us say a phobia of dogs. So the dog is a potentially fearful stimuli and basically every time you see the dog, or let us say if

you have, if the dog has bit you in the past and every time you come across dogs, there will be this, you know, aversive and implicit fear is going to be generated.

So, it seems that the amygdala kind of plays a role in learning these kind of responses. Okay? And this learning here that we are talking about is expressed more indirectly through a behavioural or a physiological response. So, you are not really telling anybody that you are afraid or anything but an autonomic arousal, a general physiological response will be generated. Let us say your heart beat will start racing up. Your, you know, palms will start sweating and your pupils will dilate. Those kinds of things will automatically happen.

Patients with amygdala damage have failed to respond, have failed to demonstrate that indirect conditioned responses for these kind of indirect conditioned responses. Say, for example, they would not hesitate in encountering stimuli such as snakes or spiders even though there is this implicit idea of that we are, you know, afraid of snakes and spiders. Further, when asked to report the parameters of fear conditioning explicitly or consciously, these patients are very good. They are, you know, they are able to demonstrate zero deficit. They are able to tell that okay this is a fear inducing situation. This is not a fear inducing situation. But say, for example, implicitly there is a difference. Implicitly, you know, they are not able to show that they actually fear the stimulus because their amygdala is damaged.

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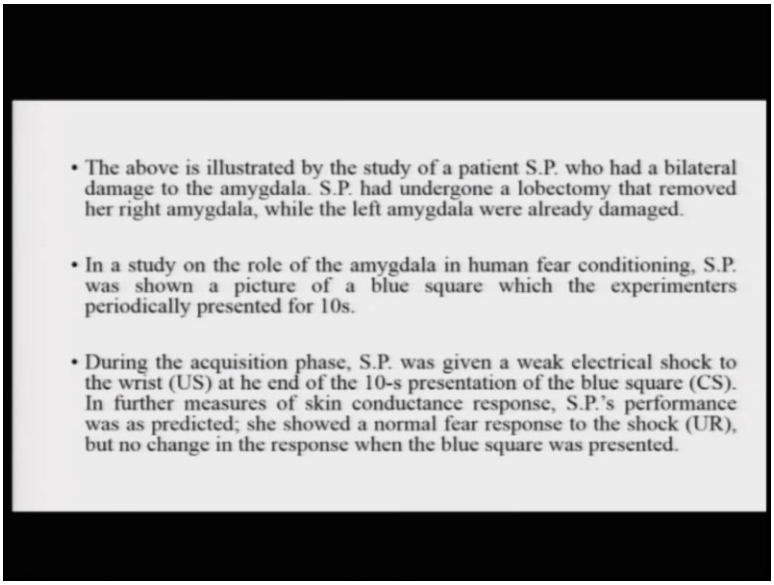

Now this can be also, I mean we have talked about this patient S.P. in the last lectures who had a bilateral amygdala damage because of the Urbach-Wiethe disease. Now she had undergone a lobectomy, no, this is a different patient, patient S.P. who also had a bilateral damage to the amygdala. Now S.P. had undergone a lobectomy that removed her right amygdala while the left amygdala was already damaged.

So, this is again a patient like S.M. that I was talking about who also has bilateral amygdala damage. Now, in a study of about the role of the amygdala in human fear conditioning, S.P. was shown a picture of a blue square in which the experimenters periodically presented, basically a blue square was periodically presented for around 10 seconds.

During the initial phase, during the acquisition phase, S.P was given a weak electrical shock to the wrist, at the end of the 10 second presentation of the blue square. So as soon as the blue square comes, it stays there for ten seconds, the participant is looking at it, by the time, it is, it is about to disappear, the patient receives a, you know, electric shock on the wrist.

Now S.P.'s performance basically, and what they, what they were measuring was they were measuring the skin conductance response which is basically a physiological response to a, you know, to all kinds of stimuli. But say, for example, they are looking for what happens in the skin conductance response in response to the presentation of the square because the square should signal, let us say for normal participants, that okay the blue square is there, I must be receiving a electric shock at some point.

Now, so in the measures of the skin conductance response, S.P.'s performance came out as predicted. She showed a normal fear response to the shock when the shock is set in but there was no change in response to the presentation of the blue square. So, although when the shock is there, she is showing the same kind of, you know, response that everybody else would show, but she is not being able to associate the presentation of the square with the electrical shock. So she is not being able to pair these two things.

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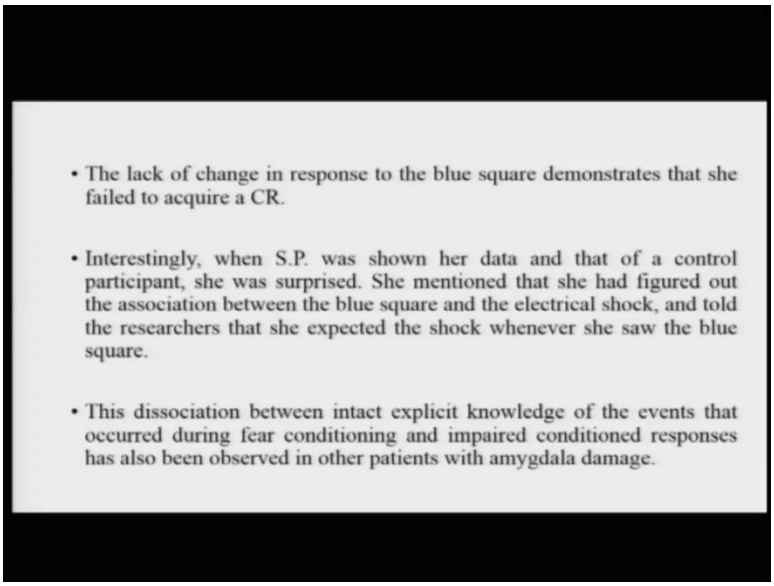

Now, this lack of change in response to the blue square demonstrates that she has failed to acquire a conditioned response. She had failed to map these two things together. Interestingly, when S.P. was shown her data and that of a controlled participant, she was actually surprised.

She mentioned that she had figured out consciously that the blue square was associated with the electrical shock and she expected that she would be shocked and she expected the shock whenever the blue square was presented. So, this is interesting. Implicitly or at the level of the physiological response, the patient's body is not showing anything. But the patient is saying that she is aware of the association between the square and the shock. So, this you can see is a dissociation between the implicit knowledge of something, the implicit knowledge that the square is, you know, signalling that the electric shock is coming and the explicit knowledge.

So, this patient has intact explicit knowledge. But she does not have intact implicit knowledge. Why does she not have the intact implicit knowledge is probably because of the amygdala damage. So, amygdala is playing a role in implicit emotional learning or implicit learning about emotional stimuli.

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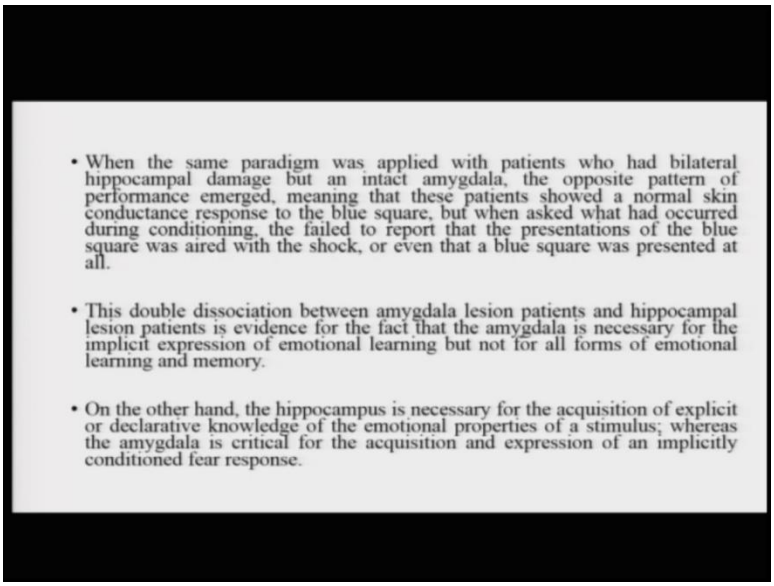

Now when the same paradigm was applied with patients who had bilateral hippocampal damage, but an intact amygdala, an opposite pattern was found. What was found is that patients showed a normal skin conductance response to the blue square but when asked, you know, normal skin conductance response to the blue square, so they actually showed change in SCR responses because they had, the participants who are able to pair the blue square with the shock. But when they were asked whether you could figure out the association between the blue square and the shock, they were unable to do so. They were sometimes unable to even report seeing a blue square. Okay? Or they had no memory of the blue square.

This again if you contrast this with the results of S.P., this kind of will give you what is called a double dissociation. We have talked about double dissociation so many times. So, this double dissociation between the amygdala lesion patients and hippocampal lesion patients, basically, is evidence for the fact that the amygdala is probably necessary for implicit expression of emotional learning but not for all forms of emotional learning.

On the other hand, the hippocampus is probably necessary for the acquisition of explicit or declarative knowledge of the emotional properties of a stimulus whereas the amygdala here again is critical for the acquisition and expression of an implicitly conditioned fear response. So, something that you are implicitly picking up is probably being picked up by the amygdala.

Something that you are sort of learning and, you know, consciously making associations, is sort of being picked up by the hippocampus.

So again, we have talked about the hippocampus in so much more detailed in the chapter on memory. This is just one more thing that you will probably need to remember.

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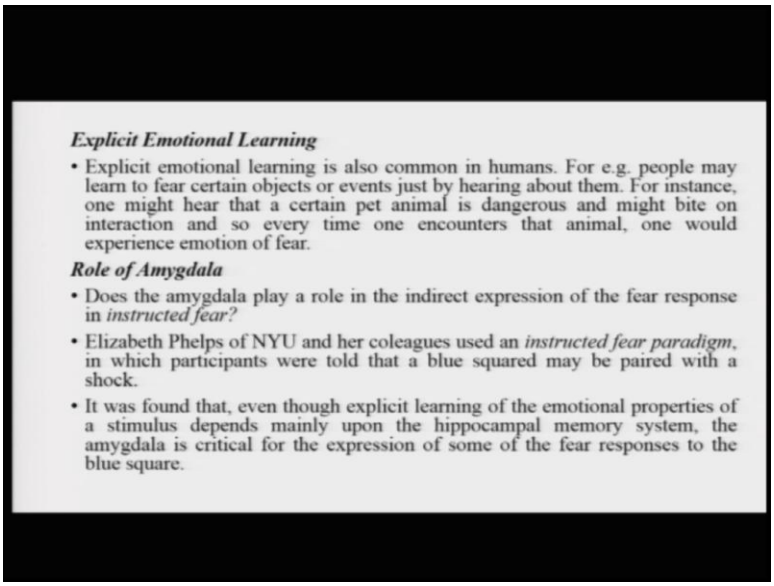

Now let us talk a little bit about explicit emotional learning. Explicit emotional learning is also common in humans. Suppose somebody tells you that, you know, you should not go and go to this place because this place is haunted and there are stories of ghosts and so many other things around this place. Every time you go to that place, you will automatically start experiencing some kind of fear. You will, you will be able to tell that you are afraid of going to that place because you have heard so many of these stories.

Does amygdala play a role in this kind of expressions as well? So, does the amygdala play a role in indirect expression of the fear response in instructed fear? When you are told that you have to be afraid of this place and then there are emotional expressions, is the amygdala going to play any role in that? That is the question, let us look at that.

Elizabeth Phelps of the New York University and her colleagues used what is called an instructed fear paradigm and, in this paradigm, what happens is that participants are told that a blue square may be paired with an electric shock. So, I'll present to you these different stimuli.

One of those stimuli will be the blue square. When we present you the blue square, there is a possibility that you will receive a electric shock.

So, when the participant is doing the experiment, you can expect that the participant should show some kind of startling response to the blue square as compared to let us say the other squares, you know, grey, green, blue, grey, green or something like that. Now it was found that even though explicit learning of emotional properties of a stimulus depends mainly upon the hippocampal memory system, the amygdala is critical for the expression of fear responses to the blue square.

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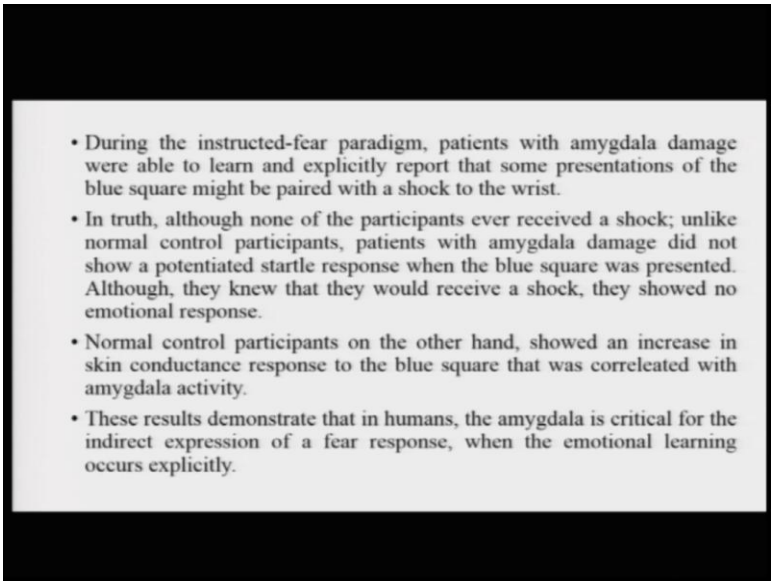

Okay, so basically, let us, let us just look at this in a little bit more detail. Let us see what has happened. During the instructed fear paradigm, patients with amygdala damage were able to learn and explicitly report that some presentation of the blue square might be paired with a shock.

In truth although none of the participants ever actually received a shock unlike the normal control part received a shock. But unlike the normal control participants, patients with amygdala damage did not show a potentiated startled response when the blue square was presented. So, their expression of this fear response is not happening.

Even though this is a case of instructed learning and the patients are consciously aware of the fact that every time a blue square come, there is a possibility, because we have been told so,

there is a possibility that we will be shocked. Unlike normal participants. Normal participants pick up this information and they express the startling response. Just like rats probably. You know, they freeze in some manner or the skin conductance response changes.

But patients with amygdala damage are unable to express this, you know, implicit fear response which is basically actually happening in a very conscious way in an instructed fear paradigm. So normal control participants on the other hand showed an increase in skin conductance response to the blue square that was corelated with amygdala activities. So, in normal participants, we can see that the amygdala is involved in getting that response out.

These results demonstrates that, in humans, amygdala is a very, very critical region for even the indirect expression of a fear response when the emotional learning offers, occurs explicitly. We have seen earlier that implicit emotional learning may be amygdala was used, explicit emotional learning also is, you know, the amygdala or say, for example, for the expression of this fear response, the amygdala is needed. Okay? Not the, not the explicit knowledge of the fear response because that we have seen that the hippocampus kind of does that.

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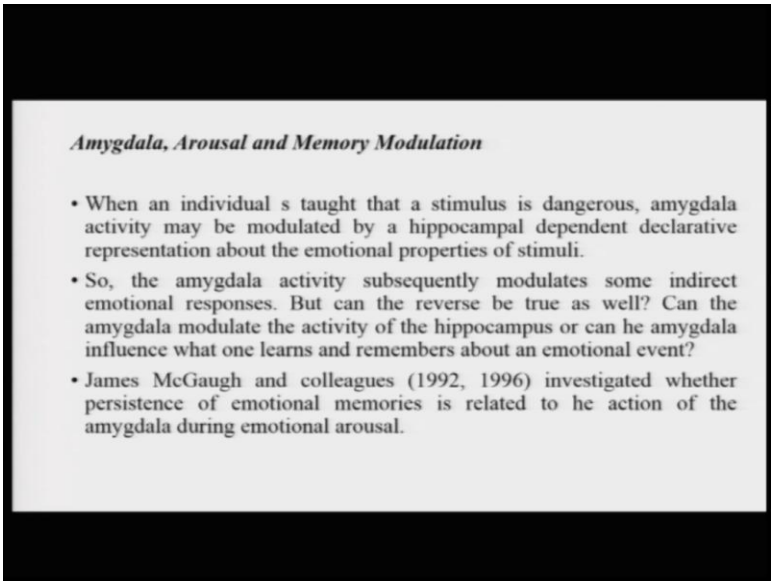

Let us talk a little bit about the amygdala, arousal and memory modulation. Now, when an individual is taught that a stimulus is going to be dangerous, typically amygdala activity may be modulated by the hippocampal dependent declarative representation about the emotional properties of the stimuli. So, the hippocampus is storing information about, okay, whether the

stimuli is dangerous, whether the stimulus is not dangerous. And so, what can happen is that the hippocampal circuitry can modulate the activity in the amygdala. That is what we are looking for.

So, the amygdala activity, then subsequently modulates, we know that the amygdala activity subsequently modulates some indirect emotional responses. Let us say, the startling response. But can they reverse be true as well? Can the amygdala modulate the activity of the hippocampus or can the amygdala influence what learn, what one learns and remembers about a particular emotional event? That is the question we want to ask about the amygdala. Now, James McGaugh and colleagues investigated whether persistence if emotional memories is related to the action of the amygdala during emotional arousal. That is the question.

Again, before I go to the next slide, what are we talking about? We are talking about, it is probable that the hippocampus kind of, because of the, you know, because of picking up the, the emotional properties of stimuli might be able to modulate the amygdala's fear response.

Is it also possible that the amygdala can modulate the, you know, the response of the hippocampus? Or say, for example, can the amygdala influence how much about a particular event we will remember or we will grasp? Okay? So, James McGaugh and colleagues investigated whether the persistence of emotional memories is related to the action of the amygdala.

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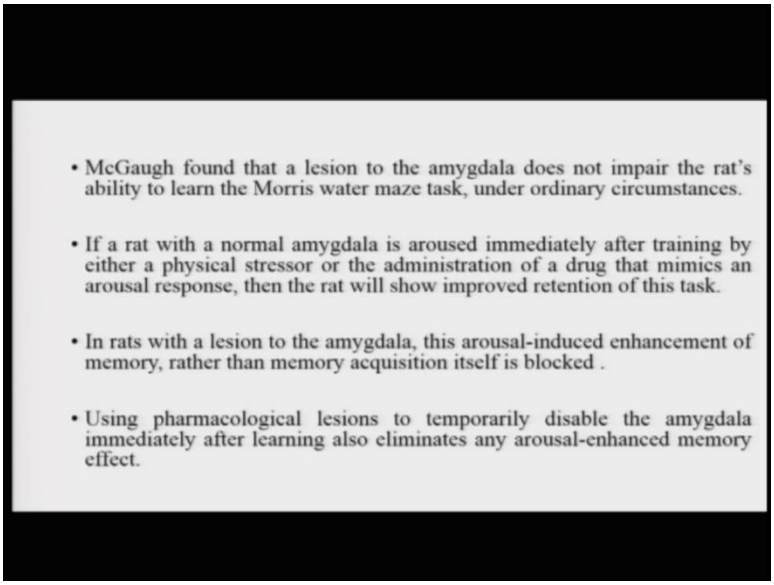

Let us see. McGaugh found that a lesion to the amygdala does not impair the rat, the rat's ability to learn the Morris water maze task. We talked about this task earlier. It is basically a task where there is a tank and, on the tank, on the surface of the tank there is this is opaque water. You cannot see through to the bottom.

So, in this opaque water there are these landmarks like windows etc. and the rat is basically dropped at various of these landmarks. And what the task of the rat is, it is to reach this platform which is underwater, is not seen from the top. Then the rat has to reach this platform so that it kind of gets saved, you know, does get saved from getting drowned. So, McGaugh actually found that if the amygdala is lesioned; it does not impair the rat's ability to learn the Morris water maze task. Okay? Under normal situations.

If, however, a rat with a normal amygdala is, if, you know, a rat with a normal amygdala is aroused immediately after training, after training by either a physical stressor or the administration of a drug that mimics an arousal response, somehow you have to get, get the rat in a slightly aroused state, then it was observed that the rat shows even a, even more improved retention of this task. So, the rats would remember the task and its outcomes for a longer time.

In rats, with lesion to the amygdala, this arousal induced enhancement of memory rather than the memory acquisition itself is blocked. So, the, the interaction between arousal and you know retention of information is sort of suffered. Okay? Now using pharmacological lesions, maybe medicinal ways to create these lesions and temporarily disabling the amygdala immediately after learning also eliminates any arousal enhanced memory effect.

So, in normal rats, arousal helps in retention of information. In rats with lesion to the amygdala, the interaction between arousal and learning of information is getting broken.

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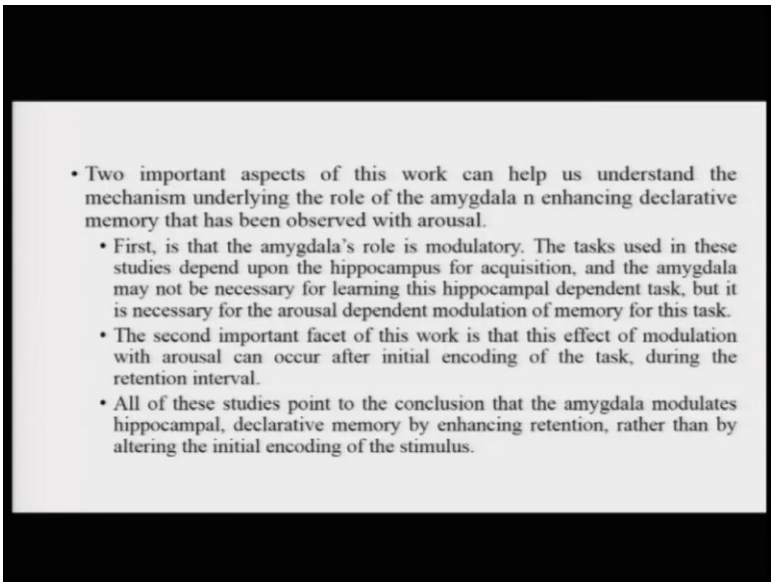

Now there is two important aspects of this work that can help us understand the mechanism that underlie the role of the amygdala in enhancing the declarative memory that has been observed in conditions with arousal. First, is that the amygdala's role is modulatory. It modulates the activation. Okay? The tasks that are used in these studies depend upon the hippocampus for acquisitions of basic learning, when there is no involvement of emotion or arousal etc., typically, just depends upon the hippocampus.

What the amygdala does is, so amygdala is not necessary for learning in this hippocampus dependent task. But it is necessary for arousal dependant modulation for memory of this task. So, for example, if the situation is emotionally aroused, if the situation is say, for example, of a pressure situation, of an intense situation, then the amygdala is there and it has typically been shown that in such kind of situations, the retention performance of rats is enhanced.

If the amygdala is damaged, this interaction between intensity, arousal and retention of information suffers. The second important facet of this work is that the effect of modulation with arousal can occur after initial encoding of the task, during the retention interval. So just one pairing is done, then the arousal is there, somehow arousal is strengthening the memory formation.

All of these studies together point, point out to the conclusion that the amygdala modulates the hippocampal, declarative memory by enhancing retention rather than by altering the initial

encoding. Because see, this retention is happening after the initial encoding. So, it is not really impacting the initial encoding. So, encoding is not getting better in any which way but the retention of information is getting better through interaction with arousal which is being done by the amygdala. So, this sort of tells you a very important aspect about the amygdala that it kind of aids emotional learning.

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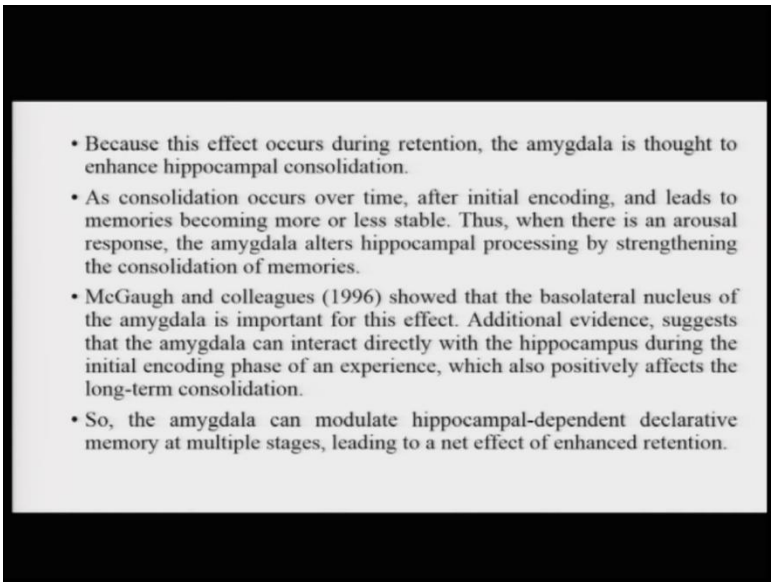

Now because this effect occurs during retention, the amygdala is thought to enhance hippocampal consolidation. Okay? So, what it is doing that it is basically helping these memories to consolidate stabilize and stay for longer durations. Okay?

Now as consolidation, as you know, we have talked about consolidation by the end of the chapter on memories, so as consolidation, you know, it occurs over time after initial encoding and leads to memories becoming more or less stable. Thus, when there is an arousal response what the amygdala probably does is it alters the hippocampal processing by strengthening the consolidation of memories.

So, that is probably the reason or the explanation for why the rats remember information better, if they do the water maze task in an aroused state. Now McGaugh and colleagues in 1996 showed that the basolateral nucleus of the amygdala is important for this effect. So, the basolateral nucleus is probably involved in this effect. Additional evidence suggests that the

amygdala can actually interact directly with the hippocampus during the initial encoding phase of an experience which also would positively affect long-term consolidation.

Because what is probably happening is that the amygdala is helping during even the encoding part. It is probably not modulating the hippocampal activity but it is providing extra information during even the encoding part which obviously must contribute towards the long-term consolidation of the retention of information.

So, the amygdala can modulate hippocampal dependent activity, declarative memory at multiple stages. At the initial encoding stage as well as in the retention stage. So, we have evidence for both kinds of, you know, modulation of the hippocampal activity by the amygdala.

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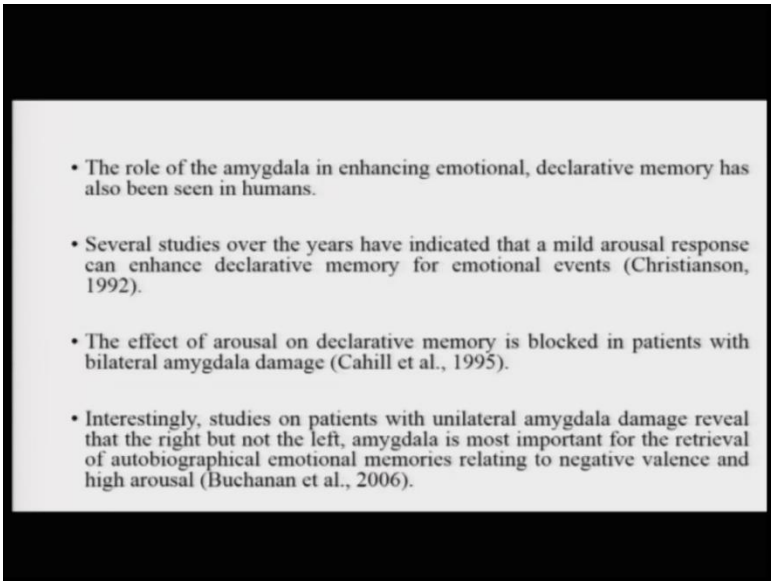

Now what is the role of the amygdala? The role of amygdala in enhancing emotional declarative memory has also been observed in humans. Several studies over the years have indicated that a mild arousal response can actually enhance declarative memory for emotional events. So, if you are aroused, if there is some degree of intensity, declarative memory might be enhanced for events that are emotional for, you know, for, you know, events that have emotional significance.

The effect of arousal on declarative memory is blocked in patients with bilateral amygdala damage. Patients who have bilateral amygdala damage, their, obviously their learning is not enhanced or there is no interaction between arousal and their declarative memory performance.

Interestingly, studies on patients with unilateral amygdala damage reveal that the right but not the left amygdala is most important for retrieval of autobiographical emotional memories, things about yourself relating to negative valence and high arousal. So, if you have had horrible experiences, if you have had something that you, you know, typically don't want to retrieve, the right amygdala is actually important in case you want to revise or in case you want to revisit those experiences.

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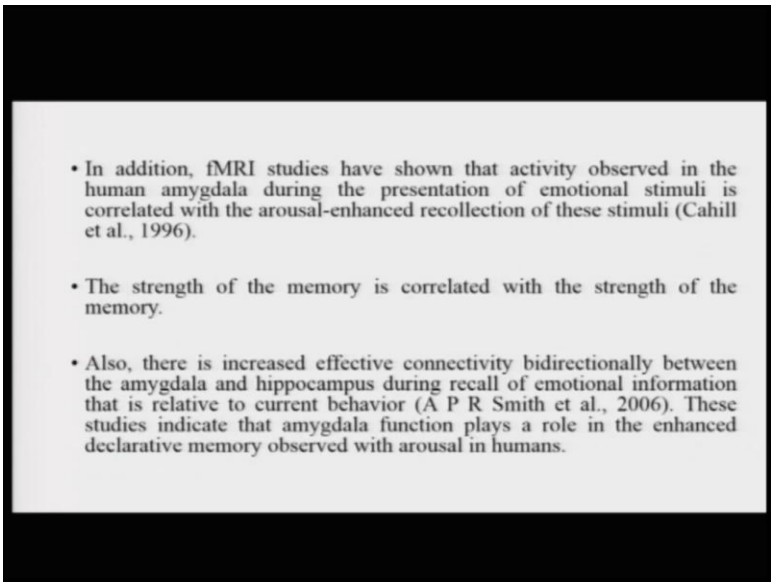

In addition, fMRI studies have shown that the activity observed in the human amygdala during the presentation of an emotional stimulus, is correlated with arousal enhanced recollection of the stimuli. So, there is again the link between arousal and amygdala and retention of information. Now the strength of the memory is correlated with, the strength of the arousal is correlated with the strength of the memory. The more aroused the individual is, the more stable the memory is. Okay?

Also, there is an increased effective connectivity bidirectionally between the amygdala and the hippocampus during the recall of information that is relative to current behaviour. So again, you can see that the hippocampus and amygdala are sort of, you know, in some sense they are collaborating to remember this, you know, emotional information.

These studies indicate that amygdala function, function does play a very important role in the enhanced declarative memory observed with arousal in humans.

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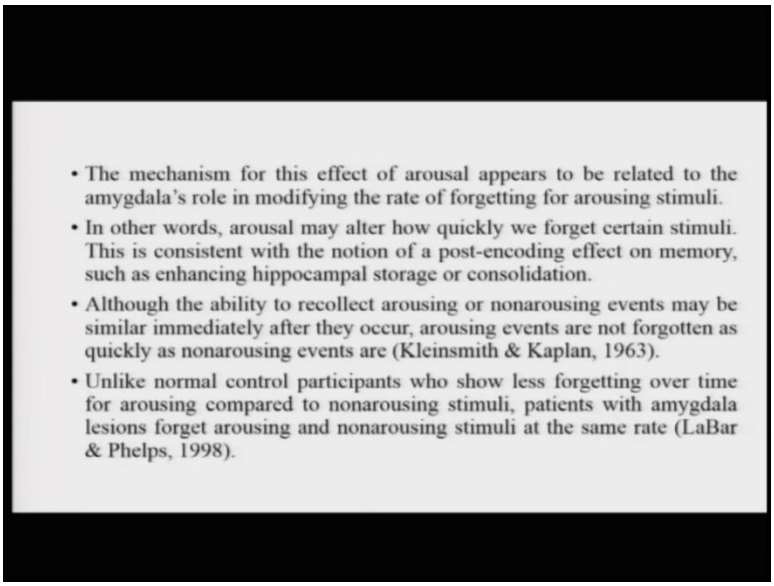

Now, what is the mechanism for this effect in humans? It appears to be related to the amygdala's role in modifying the rate of forgetting arousing stimuli as well. Okay? In other words, arousal may alter how, how quickly we forget certain stimuli.

This is consistent with the notion of a post encoding effect on memory such as enhancing hippocampal storage or consolidation. So, something that happens after the hippocampus has acquired the initial memory. Although the ability to recollect arousing or non-arousing events may be similar immediately after they occur. Arousing events are not forgotten as quickly as non-arousing events.

And this is where the amygdala probably comes in. Unlike normal control participants, who show less forgetting over town, over time for arousing compared to non-arousing stimuli, patients with amygdala lesions forget arousing and non-arousing stimuli at the same rate. Okay? So, if amygdala is damaged, they cannot differentiate between what is arousing information and what is non-arousing information.

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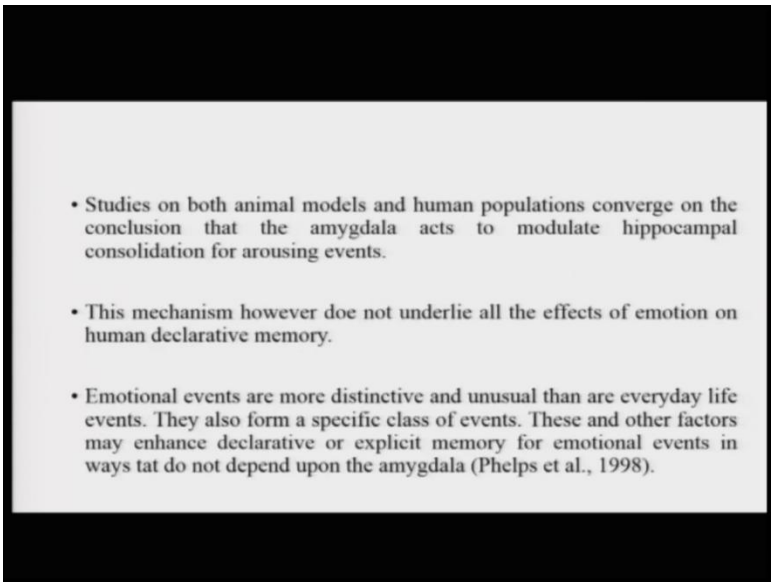

Studies on both animal models and human populations converge on the conclusion that the amygdala acts to modulate hippocampal consolidation for arousing events selectively. This mechanism however does not really underlie all the effects of emotion on human declarative memory. So, it is basically very specific to arousal related memories.

The emotional events are more distinctive and unusual than are everyday life events, we know that. They are a specific category; they are a specific class of events. And these and other factors may probably be responsible for enhancing the declarative or explicit memory for emotional events in ways that sometimes do not even depend upon the amygdala.

So, this is all about learning and role of amygdala and basically the neuro basis of emotional information learning. I will talk to you about another aspect of emotion in in the next lecture.